PLASMA HIV RNA TESTING (Updated January 10, 2011)

Plasma HIV RNA (viral load) should be measured in all patients at baseline and on a regular basis thereafter, especially in patients who are on treatment, because viral load is the most important indicator of response to antiretroviral therapy (ART) **(AI)**. Analysis of 18 trials that included more than 5,000 participants with viral load monitoring showed a significant association between a decrease in plasma viremia and improved clinical outcome *[1]*. Thus, viral load testing serves as a surrogate marker for treatment response *[2]* and can be useful in predicting clinical progression *[3-4]*. The minimal change in viral load considered to be statistically significant (2 standard deviations) is a threefold, or a 0.5 $\log_{10}$ copies/mL change.

Optimal viral suppression is generally defined as a viral load persistently below the level of detection (<20–75 copies/mL, depending on the assay used). However, isolated "blips" (viral loads transiently detectable at low levels, typically <400 copies/mL) are not uncommon in successfully treated patients and are not thought to represent viral replication or to predict virologic failure *[5]*. In addition, low-level positive viral load results (typically <200 copies/mL) appear to be more common with some viral load assays than others, and there is no definitive evidence that patients with viral loads quantified as <200 copies/mL using these assays are at increased risk for virologic failure *[6-8]*. For the purposes of clinical trials the AIDS Clinical Trials Group (ACTG) currently defines virologic failure as a confirmed viral load >200 copies/mL, which eliminates most cases of apparent viremia caused by blips or assay variability *[9]*. This definition may also be useful in clinical practice. (See **Virologic and Immunologic Failure**.)

For most individuals who are adherent to their antiretroviral (ARV) regimens and who do not harbor resistance mutations to the prescribed drugs, viral suppression is generally achieved in 12–24 weeks, even though it may take longer in some patients. Recommendations for the frequency of viral load monitoring are summarized below.

- **At Initiation or Change in Therapy.** Plasma viral load should be measured before initiation of therapy and preferably within 2–4 weeks, and not more than 8 weeks, after treatment initiation or after treatment modification **(BI)**. Repeat viral load measurement should be performed at 4–8-week intervals until the level falls below the assay's limit of detection **(BIII)**.
- **In Patients Who Have Viral Suppression but Therapy Was Modified Due to Drug Toxicity or Regimen Simplification.** Viral load measurement should be performed within 2–8 weeks after changing therapy. The purpose of viral load monitoring at this point is to confirm potency of the new regimen **(BIII)**.
- **In Patients on a Stable ARV Regimen.** Viral load should be repeated every 3–4 months or as clinically indicated **(BII)**. Some clinicians may extend the interval to every 6 months for adherent patients who have suppressed viral loads for more than 2–3 years and whose clinical and immunologic status is stable **(BIII)**.

Monitoring in Patients with Suboptimal Response. In addition to viral load monitoring, a number of additional factors, such as adherence to prescribed medications, altered pharmacology, or drug interactions, should be assessed. Patients who fail to achieve viral suppression should undergo resistance testing to aid in the selection of an alternative regimen, as discussed in **Drug Resistance Testing** and **Virologic and Immunologic Failure** **(AI)**.

References

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